SUXAMETHONIUM SENSITIVITY — YET ANOTHER CASE

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SUMMARY
Abnormal variants of plasma cholinesterase are a rarity in this region and to date there is only one reported case of suxamethonium sensitivity in a Malaysian population. We now report a case of a Malaysian Chinese patient who received suxamethonium, developed prolonged apnoea and on investigation found to be a homozygote for the silent gene. His family was screened for abnormal variants of plasma cholinesterase. The results are discussed.

INTRODUCTION
Prolonged apnoea following the administration of short acting muscle relaxant suxamethonium (scoline) due to abnormal variants of plasma cholinesterase is a well documented entity. To date there is only one reported case of suxamethonium sensitivity in a Malaysian population.

CASE REPORT
A 27 year old Malaysian Chinese was admitted for treatment of appendicitis. Pre-anaesthesic examination revealed a normal patient and all investigations were within normal limits. Anaesthesia was induced with 250 mg Thiopentone and relaxation obtained with 100 mg suxamethonium. Patient was intubated and ventilated with nitrous oxide-oxygen mixture. Pentazocine 30 mg was given for analgesia. No other relaxant was given as the patient did not recover from the first dose of suxamethonium. At the end of the operation there was no evidence of spontaneous respiration. Patient was transferred to the intensive care unit and ventilated with 50% nitrous oxide-oxygen mixture. Full muscular power was regained at the end of four and a half hours. Peripheal nervestimulator indicated a depolarisation neuromuscular block. Blood taken for estimation of plasma cholinesterase showed a low level of the enzyme. Patient’s family was screened consequently and the results are given below.

**TABLE I**

<table>
<thead>
<tr>
<th>Indiv­idual</th>
<th>Serum Cholines­terase values (ChE. No)*</th>
<th>Dibu­caine No.</th>
<th>Phenotype</th>
</tr>
</thead>
<tbody>
<tr>
<td>I₁</td>
<td>49</td>
<td>80</td>
<td>Normal homozygote</td>
</tr>
<tr>
<td>I₂</td>
<td>39</td>
<td>60</td>
<td>Heterozygote</td>
</tr>
<tr>
<td><strong>II₁</strong></td>
<td>1</td>
<td>0</td>
<td>Abnormal homozygote</td>
</tr>
<tr>
<td>II₂</td>
<td>67</td>
<td>72</td>
<td>Normal homozygote</td>
</tr>
<tr>
<td>II₃</td>
<td>66</td>
<td>67</td>
<td>Normal homozygote</td>
</tr>
<tr>
<td>II₄</td>
<td>61</td>
<td>67</td>
<td>Normal homozygote</td>
</tr>
<tr>
<td>III₁</td>
<td>61</td>
<td>75</td>
<td>Normal homozygote</td>
</tr>
<tr>
<td>III₂</td>
<td>68</td>
<td>72</td>
<td>Normal homozygote</td>
</tr>
</tbody>
</table>

**PROPOSITUS**
* ChE NO: Cholinesterase Number (△ pH units/Hour x 100).
studied. Plasma cholinesterase estimation was done according to the electrometric method, and dibucaine numbers were determined according to the spectrometric method.

RESULTS

The results are shown in Table I. The serum cholinesterase values are reported in \( \Delta \) pH units/Hour x 100 (cholinesterase number). The table also shows the probable phenotypes.

The propositus and his family are shown in Fig. 1.

DISCUSSION

The results of our study show that the propositus is a homozygote for the silent gene, with a very low level of enzyme activity and a dibucaine number 0. His children, father, brother and sister have normal enzyme activity and normal dibucaine number. His mother was found to be heterozygote. The exact genotyping could not be done due to the non-availability of fluoride numbers. All the individuals in our study were in good health. The propositus and his mother were given warning cards (Fig. 2) to be shown to the anaesthetist should an operation be necessary.

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REFERENCES

